The bilateral limb of her consciousness was briefly followed by a slight band-like headache. Surrounding this, there were several additional neurological abnormalities, and the patient complained of several physical disturbances. The left temporal region showed a few brief waves, which generalized, and there were multiple amnestic episodes. The patient was in the alert state during this period.

The CT scan showed a rounded area of contrast enhancement in the temporal region. The patient was not aware of the changes in consciousness. However, the EEG showed a high level of activity, and there were no signs of focal lesions. The patient was in the alert state during this period.

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sensory disturbances closely resembling an ischaemic process.

Intra-arterial embolism of an aneurysmal sac thrombosis is considered a possible cause of transient ischaemic deficit or completed stroke in patients with unruptured, intracranial aneurysms. In our case the demonstration by MRI of intraluminal thrombus material inside the aneurysm gives support to the view that thrombi may have dislodged from the clot and embolised into the distal vessels. Alternatively the thrombosis process may extend from the aneurysm and involve the lumen of an artery arising from the basilar artery and so cause ischaemia. On the other hand, it seems unlikely that platelet embolisation from an extracranial source was the cause because the patient had no stenosis or ulceration, in these vessels; neither a cardiac source, systemic hypotension, nor underlying conditions predisposing to hypercoagulability were identified.

Regardless of the physiopathological mechanisms, the case reported shows that the PSS syndrome can occur secondarily to a basilar aneurysm. These aneurysms provide another example of the many potential aetologies of lacunar syndromes.

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Figure Top—contrast-enhanced CT scan showing round area of increased density in upper interpeduncular fossa. MIP—MRI (Sagittal, T2 weighted) showing aneurysm extending from upper pons to inferior aspect of third ventricle compressing left cerebral peduncle. Bottom—MRI (Axial, T2 weighted) showing hyperintense images within aneurysm suggesting clot inside its lumen.

have been found at necropsy or during neurological evaluation for other reasons. In our case the association of ischaemic attacks with an aneurysm in the appropriate vascular distribution without any evidence of other abnormalities of the CNS strongly suggests a causal relation rather than an incidental finding. Transient neurological disorders resembling TIAs or reversible ischaemic neurological deficits have been described in patients with intracranial unruptured aneurysms, but although an ischaemic process was suspected in all cases, the exact pathophysiological mechanisms of these disturbances remain obscure. Focal neurological events have been attributed to the compressive effects of large intracranial aneurysms. The abrupt onset of symptoms may occur because enlarging aneurysms acutely obstruct a penetrating branch of the basilar artery within the confines of the aneurysm. A mechanism of direct compression on the adjacent cerebral structures seems unlikely in our patient as he showed transient

removed incompletely. The other tumour was bean-sized and located under the left optic nerve; this tumour was removed completely. The pathologists who were consulted had difficulty in making a definite diagnosis: reticulocarcinomatous and malignant epithelial tumour were suggested. An extensive search for tumour did not reveal any. The patient was treated with radiotherapy followed by systemic chemotherapy (12 courses of cyclophosphamide and vincristin). The whole brain irradiation was 23-54/Gy, treatment coned down to (para) sellar areas was given up to 60 Gy. Special attention was paid to include the suprasellar area for up to 3 cm, and the whole of the sphenoid; the clivus was only partly irradiated. In 1979 a CT of the brain revealed nothing abnormal. In 1988, the patient was found in an unempt condition and was admitted in a delirious state and with neck rigidity. Body temperature was 34°C, T4, TTA index and T3 uptake were 65 nmol/l (N = 80–150), 55 nmol/l (N = 70–170) and 0-66 nmol/l (N = 0-95–1-20) respectively. Serum TSH was normal. CT protein was 20 gr/l, cell count was normal and no pathological examination of the fluid did not identify tumour cells.

The patient’s condition improved with general supportive treatment and levothyroxin and dexamethasone. CT and MRI of the brain and spinal cord identified a mass located anterolateral to the pons and medulla oblongata, mainly on the right side and extending down to the sixth cervical vertebra. Intraspinal lesions were also present in the high and mid thoracic regions. To obtain a tissue diagnosis, stereotactic biopsies were taken under local anaesthesia. Smears made during this procedure suggested a pituitary adenoma. The patient died suddenly on the third postoperative day. At necropsy the immediate cause of death appeared to be aspiration of food. No other abnormalities were found outside the central nervous system. Grossly as well as microscopically no abnormalities were found in the para- and suprasellar region, pituitary fossa or sphenoïd sinuses. The pituitary tumour tissue was found in the lesions shown by CT and MRI. Histopathologically, the tumour consisted of sheets of medium-sized epithelial cells with round or oval nuclei surrounded by faintly stained amorphous cytoplasm. Mitotic figures were rare and nuclear polymorphism was inconspicuous. Immunoreactions for keratin and NSE were positive, and for S-100 weakly positive. Very few cells reacted positively for chromogranin A. Between 10-25% of the cells reacted strongly for prolactin, less than 10% for growth hormone. Ultrastructural examination of stereotactic biopsy showed a few 100-200 nm dense granules, often surrounded by a membrane (figure). The histological appearance of the specimens obtained from 1975 and 1988 were identical; the paraffin blocks from 1975 were not available for further study. The diagnosis herefore was of an ectopic parasellar pituitary adenoma, with a normal intrasellar pituitary producing leptomeningeal seeding.

The persistence of etoposide or paraspinal or paraseellar pituitary adenoma with a normal intrasellar pituitary gland is not surprising in view of the recognition by Horii of "supraadenohypophyseal" glands in the leptomeninges surrounding the pituitary stalk and infundibulum. These cells were found in all foetuses examined and in 75% of adult necropsies. In the present case, between


Ectopic paraseellar pituitary adenoma with subarachnoid seeding

There are very few descriptions of an ectopic supra- or paramesencephalic adenoma in a patient with a normal intrasellar pituitary gland. In one case the tumour originated in the parasellar region, and in a third case the tumour arose either in the sphenoid or in the parasellar region. We report a further case of ectopic paraseellar paraneural adenoma which was complicated by subarachnoid seeding 13 years after initial treatment by craniotomy, radiotherapy and systemic chemotherapy.

In 1975 a 45 year old man who had been ipotent revealed. Cranial and cervical CT scans disclosed two mucoid tumours. One was pea-sized, seemed to infiltrate into the left oculomotor nerve near its entrance into the cavernous sinus, and was
Transient pure sensory strokes in patient with aneurysm of rostral basilar artery.

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